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Original Article

Effect of repetitive peripheral magnetic stimulation of the common fibular nerve on the soleus muscle Hoffmann reflex

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Abstract. [Purpose] This study aimed to investigate the effects of repetitive peripheral magnetic stimulation of the common fibular nerve on the modification of neural circuit function as measured through the soleus muscle Hoffmann reflex. [Participants and Methods] Twenty-four healthy adult males were randomly and equally divided into the magnetic stimulation (experimental) and control groups. The Hoffmann reflex of the soleus muscle was analyzed before and after 10 min of repetitive peripheral magnetic stimulation for the experimental group and before and after 10 min of rest for the control group. The averages of the values for the maximum amplitude and latency of the Hoffman reflex across twenty repetitions were recorded and compared. [Results] The Hoffmann reflex amplitude decreased following stimulation in the experimental group, and significant variations were observed between the experimental and control groups. [Conclusion] The change in the Hoffmann reflex amplitude may have been caused by the magnetic stimulation to I-a sensory fibers on the common fibular nerve, suggesting that magnetic stimulation induces reciprocal inhibition of motor neurons through synapses in the spinal cord.
Key words: Repetitive peripheral magnetic stimulation, Soleus Hoffmann reflex, I-a reciprocal inhibition

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INTRODUCTION

In central nervous system disorders, such as cerebrovascular diseases and spinal cord injuries, combining physical therapy with electrical or magnetic stimulation has been shown to enhance motor function recovery. The first report on Paired Associative Stimulation (PAS), which synchronizes transcranial magnetic stimulation (TMS) with peripheral nerve electrical stimulation, was presented by Stefan et al.[1)], highlighting its greater utility compared to individual physical therapy or electrical stimulation for peripheral nerves. Tomaru[2)], Weingarden et al.[3)], Sheffler et al.[4)], and Popovic et al.[5)] demonstrated that performing TMS simultaneously with voluntary movement or electrical stimulation can enhance the excitability of the corticospinal pathway to promote neuroplasticity in the brain and spinal cord related to functional recovery.

In central nervous system disorders such as strokes, motor paralysis of agonist muscles and spasticity of antagonist muscles are common, and these factors in combination inhibit motor function recovery. Therefore, preventing these factors is important in physical therapy. The mechanisms for inhibiting spasticity include the influence of input stimuli on the agonist muscles via peripheral nerves, such as I-b inhibition[6)], and I-a reciprocal inhibition of the antagonist muscles[7)]. The effects of functional electrical stimulation on motor paralysis involve increased recruitment because of firing in the spinal interneuron area, and the influence on nervous system cell-plasticity through neural function modifications, such as an increase in spinal Renshaw cell activity and its recurrent inhibition[8–10)].

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In recent years, peripheral magnetic stimulation (PMS) therapy has gained attention as an alternative to electrical stimulation for stimulating peripheral sites. PMS generates eddy currents through electromagnetic induction within the body, promoting muscle contraction[11, 12]. The magnetic stimulation can stimulate without activating pain receptors (Aδ, C fibers) in the skin, resulting in less discomfort or pain during stimulation[13].

In 2013, Beaulieu et al.[14] reviewed the literature on the effects of magnetic stimulation in healthy adults, as well as stroke and spinal cord injury patients. Furthermore, Beaulieu et al.[15] provided a review in 2015 on the therapeutic effects of magnetic stimulation devices for pain and peripheral nerve disorders. These reviews highlighted the enhancing effects of magnetic stimulation on motor function in individuals with disabilities (changes in EMG, muscle strength, ADL) and its inhibitory effects on muscle tone (Ashworth Scale, tendon reflexes)[14], as well as improvements in pain (VAS and Algometer numerical improvements) and peripheral nerve disorders[15]. Kagaya[16] summarized previous literature on the clinical applications of PMS and provided explanations on stimulation parameters for PMS and the development of coils for stimulating suprahyoid muscles[16]. In addition, other reports[17–21] on magnetic stimulation have described the possiblities[20] or difficulties[19] in stimulating the I-a sensory nerves and spinal cord circuits using magnetic stimulation. Consequently, few reports elucidate the mechanisms by which magnetic stimulation or repetitive peripheral magnetic stimulation (rPMS) affect spinal cord circuits.

Assuming that different magnetic stimulation foci (e.g., nerve point, motor point) and stimulation intensities may affect spinal cord circuits, we explored ways to modulate the neural circuit function while capturing changes in spinal nerve mechanisms through peripheral nerves and muscles under rPMS. This study reports how the application of rPMS to the common fibular nerve affects the soleus muscle Hoffman reflex (H-reflex, an index of excitability of the motor neuron pool in the spinal cord).

PARTICIPANTS AND METHODS

Twenty-four healthy adult males were recruited as participants and randomly assigned to the magnetic stimulation group (rPMS group, N=12, age 18–22 years, mean ± SD height: 172.3 ± 5.1 cm, weight: 64.9 ± 8.1 kg) and control group (N=12, age 18–22 years, height: 169.0 ± 6.2 cm, weight: 61.9 ± 8.4 kg).

Prior to study initiation, the participants were informed of the study's purpose and details, including its benefits and risks, and advised that their personal information would be protected and that they could refuse to participate or withdraw from the study. They were recruited after providing informed consent for participation. This study was approved by the research ethics committee of the Tokyo International University (approval number: 21-2).

The limb position for recording the soleus muscle H-reflex (Fig. 1) was the supine position, with the participant's hip and knee joints flexed at 10° and the ankle joint in a neutral position, and with limbs secured to the measurement platform with a belt. Both the rPMS and control groups started with 10 min of rest in the supine position. Subsequently, the rPMS group underwent H-reflex measurements before and after 10 min of magnetic stimulation intervention, and the control group had measurements taken before and after 10 min of rest. The H-reflex measurement system used for the soleus muscle was an electromyogram and evoked potential testing device (Neuropack MEB-9400, Nihon Kohden, Tokyo, Japan). The stimulation for evoked potentials was applied to the tibial nerve in the right popliteal fossa, with the anode fixed at the center of the patella and the cathode fixed to the tibial nerve in the popliteal fossa for electrical stimulation. The stimulus current had a pulse width of 1 ms and frequency of 0.3 Hz; the stimulation intensity was gradually increased from 0 mA until the H-reflex

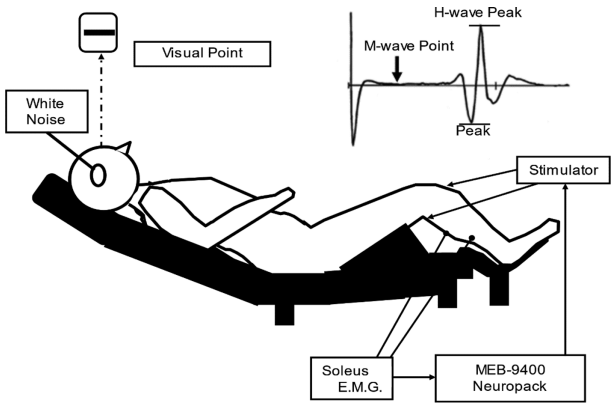

Fig. 1. Block diagram of experiment.
H-wave: Hoffmann Reflex, index of excitability of the motor neuron pool in the spinal cord.
M-wave: Index of excitability of α-motor neurons.
Soleus E.M.G.: Soleus electromyogram.

appeared, followed by a further increase until the M-wave appeared. The stimulation intensity at the point where the M-wave disappeared was recorded as the stimulus intensity (Fig. 1).

The H-reflex data were recorded from 1 min after the initiation of current stimulation once the H-reflex amplitude stabilized. The data included the peak-to-peak maximum amplitude of the H-reflex for 20 repetitions and latency at the onset of the H-reflex, which were then averaged.

The magnetic stimulation in the rPMS group was delivered using a repetitive skeletal muscle magnetic stimulator (Pathleader, IFG Co., Ltd., Sendai, Japan) to the area just below the right fibular head, at a maximum flux change rate of 11–20 kT/s, pulse width of 350 μs, and frequency of 50 Hz, administered below the pain threshold (mean intensity 84.2 ± 10.8%) for 10 min. The frequency of magnetic stimulation was set at 1 per 10 s, with a stimulus duration of 2 s and rest period of 8 s, resulting in 60 stimulations over the 10 min period.

During H-reflex measurements, participants in both groups were instructed to keep their eyes open, fixate on a focal point, and listen to white noise through headphones as the auditory stimulus.

To compare the interactions before and after the intervention for H-reflex maximum amplitude and H-reflex onset latency in the rPMS and control groups, a two-way analysis of variance (ANOVA) was performed (5% significance level) using SPSS Version 24 for Windows (IBM Corp., Chicago, IL, USA).

RESULTS

The results of the H-reflex amplitude measurements are presented in Table 1 and Fig. 2. The two-way ANOVA for H-reflex amplitude showed a significant interaction ($p<0.05$) before and after the intervention in both the rPMS and control groups.

The results of the two-way ANOVA for H-reflex latency (Table 1) did not show a significant interaction between groups.

Table 1. Mean Hoffmann reflex (H-reflex) maximum amplitude and latency in each group

	rPMS group		Control group		ANOVA
	Pre	Post	Pre	Post	
H-reflex maximum amplitude (mA)					
Mean ± SD	10.1 ± 4.8	9.2 ± 5.2	6.7 ± 3.3	7.1 ± 3.4	*
H-reflex latency (msec)					
Mean ± SD	28.9 ± 1.8	29.6 ± 1.9	28.7 ± 1.7	29 ± 1.8	
Intensity of H-reflex electric current stimulation (mA)					
Mean ± SD	11.1 ± 6.1		14.1 ± 6.5		

*$p<0.05$.
rPMS: repetitive peripheral magnetic stimulation; ANOVA: two-way analysis of variance; SD: standard deviation.

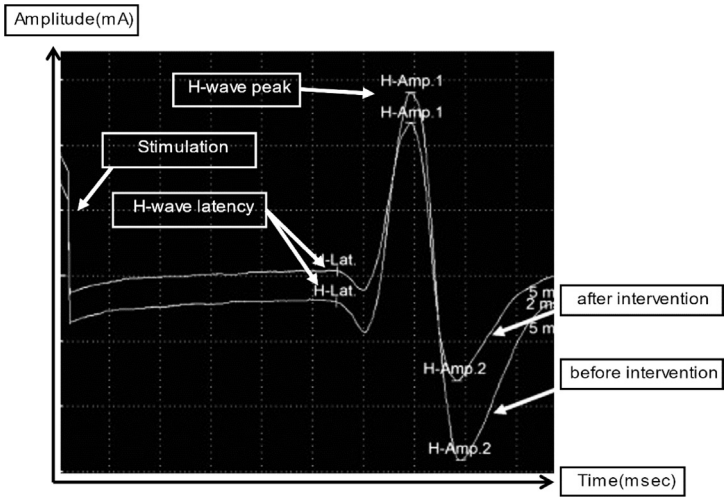

Fig. 2. H-wave of before and after intervention.

DISCUSSION

Changes in the H-reflex amplitude after magnetic stimulation in the rPMS group showed a significant interaction ($p<0.05$) before and after intervention in both the rPMS and control groups. This result was thought to influence the I-a reciprocal inhibition for the common fibular nerve by magnetic stimulation.

Delwaide[22] reported a significant reduction of the H-reflex in the Achilles tendon of spastic paralysis patients when subjected to continuous vibrational stimulation at around 100 Hz. The inhibition of the H reflex was reported due to stimulation of the I-a sensory fibers of the homonymous muscle that is sensitive to vibrational stimulation, resulting in ascending stimulation and presynaptic inhibition of the spinal cord[23]. In this vibrational stimulation, the effects of post-activation depression[24, 25], such as inactivation of the Ca^{+} channels at synaptic terminals and a decrease in neurotransmitter release, were thought to occur owing to the frequent firing of I-a inputs. In our study, we applied magnetic stimulation to the common fibular nerve, which innervates the heteronymous muscle against the soleus muscle, unlike the inhibition reported by Desmedt[22, 23] for the homonymous muscle. Crone et al.[7] measured the H-reflex during voluntary dorsiflexion movements of the ankle joint and reported the involvement of long-latency inhibition due to I-a reciprocal and presynaptic inhibitions based on changes in the excitatory level of the spinal cord. Hirabayashi et al.[26, 27] reported three inhibitory pathways involved in smooth joint movements between antagonistic muscles. These pathways include (1) Two-synaptic I-a reciprocal inhibition, where inhibitory interneurons mediate the direct synaptic connection from the afferent I-a fibers of the agonist muscles to the spinal anterior horn cells of the antagonist muscle, inhibiting the antagonist muscle. In addition, afferent I-a fibers from the agonist muscles connect to the terminals of the afferent I-a fibers of the antagonist muscle via primary afferent depolarizing interneurons, acting as presynaptic inhibition; (2) Short-latency inhibition (D1 inhibition), and 3) Long-latency inhibition (D2 inhibition)[27]. In these reports, it was mentioned that reciprocal inhibition suppressed excessive antagonist muscle contractions during movement, enabling smooth and coordinated movements. Kagamihara et al.[28, 29] described reciprocal inhibition as the phenomenon of inhibiting the antagonist muscle accompanying the excitation of the agonist muscle during movement. They explained the relationship between the mechanical contraction of the agonist muscles and stretch reflex of the antagonist muscle and reported the existence of the two-synaptic I-a circuit as the mechanism involved in this process. Hirabayashi et al.[26, 27] and Kagamihara et al.[28, 29] have mentioned that the impairment of the I-a reciprocal inhibitory circuit's activity is related to paralysis of the dorsal flexor muscles and an abnormal increase in muscle tone of the antagonistic plantar flexor muscles while walking, in patients with upper motor neuron disorders. They discussed ways to enhance reciprocal inhibition to improve these conditions and mentioned that peripheral stimulation such as Patterned Electrical Stimulation or repetitive passive movements might be more effective in producing after-effects compared to brain stimulation alone[27]. Fok et al.[30] reported on the effectiveness of using the motor point as a site for peripheral neuromuscular stimulation in the PAS, a method that synchronizes peripheral nerve electrical stimulation with TMS of the primary sensory motor cortex. Fok et al.[30] and Nakagawa et al.[31, 32] reported on the differences between motor point and nerve site stimulations as methods for peripheral neuromuscular electrical stimulation. These reports[30–32] mentioned that nerve site stimulation allows for the stimulation of I-a sensory nerves, whereas this would be difficult at the motor point. They discussed that electrical stimulation at the motor point has the potential to travel peripherally from the stimulation site or reach the synapses of spinal motor neurons by retrograde conduction along the axons of α-motor fibers. The studies conducted by Fok et al.[30], Nakagawa et al.[31, 32], and others primarily focused on electrical stimulation and reports related to active or passive movement, and few studies have investigated the mechanism of rPMS effects.

In our study, the H-reflex of the soleus muscle decreased after rPMS application on the common fibular nerve. We found that rPMS could have affected the spinal cord neural circuits through peripheral nerves. The observed phenomenon has been influenced by I-a reciprocal inhibition. However, reports by Zhu et al.[19] indicated that measuring the H-reflex using a single magnetic stimulus might underestimate it. This report mentioned that sensory nerve stimulation with a single magnetic stimulus has a short stimulation width (duration), making it difficult to stimulate sensory nerves effectively. Martin et al.[17] conducted rPMS with varied stimulation sites on the muscle belly of the soleus muscle and measured the H-reflex and M-wave as indicators of spinal cord excitability before and after the intervention. The results showed no significant changes in the H-reflex with rPMS applied to the muscle belly. Nito et al.[21] measured motor-evoked potentials (MEPs) of the hand extensor muscle using TMS with the H-reflex and M-wave as indicators of spinal cord excitability before and after rPMS on the muscle belly of hand extensor muscles in healthy adults. In both studies by Martin et al.[17] and Nito et al.[21], the H-reflex, an indicator of spinal cord excitability, did not show significant changes.

These findings suggested that in future it would be necessary to advance basic research to understand the mechanisms by which rPMS affects spinal cord neural circuits through peripheral nerves and muscles, such as the differences between single-pulse and repetitive magnetic stimulation, relationship between stimulation sites (nerve sites and muscle belly), possibility of stimulating I-a sensory neurons with rPMS, I-a reciprocal inhibition, and the facilitation of α-motor neurons.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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